Outcome of Splenectomy for Thrombocytopenia Associated With Systemic Lupus Erythematosus

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Objective: To determine the efficacy of splenectomy for treating thrombocytopenia associated with systemic lupus erythematosus (SLE).

Summary Background Data: The role of splenectomy has been controversial in this patient population.

Methods: Between 1975 and 2001, 25 consecutive adults with SLE underwent splenectomy specifically for thrombocytopenia. Surgical indications, operative mortality and morbidity, and hematological outcomes were followed in both the short-term (first 30 days) and the long-term (last recorded platelet count, last contact, or death). Response to splenectomy was rated as: complete (CR: platelets $\geq 150 \times 10^9$/L for at least 4 weeks), partial (PR: platelets $50-149 \times 10^9$/L for at least 4 weeks), or none (NR: platelets $< 50 \times 10^9$/L at all times). Relapse occurred if platelets fell below 50×10^9/L after CR or PR.

Results: Indications for splenectomy included: thrombocytopenia refractory to (64%), dependent on (20%), or patient intolerance of (16%) medical treatments. Perioperative mortality was 0% and morbidity was 24%. After a median of 9.5 years, 9 patients (36%) had died, with only 1 death being secondary to bleeding. Early partial or complete response rate to splenectomy was 88%. After a median follow-up of 6.6 years, 16 (64%) patients had sustained complete or partial response without relapse. Eight (32%) of these patients required adjunctive medical therapy, whereas the other 8 (32%) did not. The remaining 9 (36%) patients relapsed, but 5 (20%) of the 9 patients were subsequently salvaged to at least partial response with further treatments. The overall PR or CR to splenectomy combined with medical therapy was 84%.

Conclusion: Splenectomy should be considered safe and efficacious for thrombocytopenia associated with SLE.

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Systemic lupus erythematosus (SLE) is a chronic systemic autoimmune disease. Between 8% and 20% of the patients with SLE may be affected by thrombocytopenia.[1,2] Thrombocytopenia in SLE not only compromises the quality of life, but also increases the risk of mortality.[3,4] Effective treatments for thrombocytopenia associated with SLE are, therefore, important clinically.

Thrombocytopenia associated with SLE is immune-mediated.[5,6] Antiplatelet antibodies are demonstrable in nearly 78% of patients with SLE.[7] Immune-complex coating of platelets leads to their early destruction by the complement system.[1,8,9] Although the exact mechanism of platelet destruction is unknown, the spleen has been implicated either as the source of antiplatelet antibodies or as the sequester of sensitized platelets.[10,11] Numerous immune-modulating medications have been used to treat thrombocytopenia of SLE and have included corticosteroids,[9] danazol,[12] intravenous immunoglobulin (IVIG),[13–15] and immunosuppressive and antineoplastic agents.[16,17] Despite their reported success in correcting thrombocytopenia, their use is often limited by significant clinical side effects. Thus, the best treatment of thrombocytopenia of SLE has not been defined.

Idiopathic thrombocytopenic purpura (ITP) is another disease with immune-mediated thrombocytopenia,[18,19] responsive to many of the same immune-modulating medications that treat thrombocytopenia of SLE. Splenectomy has become a well-established treatment, in addition to, or even preferable to, medical therapy for patients with ITP,[20] with remission rates between 75% and 94%.[21] Whether splenectomy is similarly effective for thrombocytopenia associated with SLE is unclear, despite several single-institution reports.[1,10,22,23] Several issues remain controversial: 1) the safety of operating on patients with SLE who may have multiorgan system diseases; 2) the durability of clinical response to splenectomy; and 3) the incidence of relapse and the salvage treatments for recurrent thrombocytopenia.

We, therefore, reviewed our experience with splenectomy performed specifically for thrombocytopenia associated with SLE over the last 26 years. Operative mortality and morbidity rates, long-term survival and complications, early

and late hematological responses, and relapse rates were assessed as outcomes.

METHODS

After approval by the Institutional Review Board, institutional medical and surgical index databases were used to identify: 1) all adult (>16 years old) patients evaluated at Mayo Clinic, Rochester, MN, for SLE between 1975 and 2001; and 2) all adult patients who underwent open or laparoscopic splenectomy during the same time period. These 2 sets of databases were cross-referenced, and all patients with SLE who underwent splenectomy were culled. Only those who had splenectomy for the specific indication of thrombocytopenia were included in this study.

The diagnosis of SLE was based on current criteria set by the American Rheumatism Association.[24,25] Thrombocytopenia was defined as platelets $< 150x10^9$/L and severe thrombocytopenia as platelets $\leq 50 \times 10^9$/L. Patients with concomitant cytopenias (eg, anemia in Evans syndrome) were excluded. We included patients who carried the initial diagnosis of ITP and later satisfied the diagnostic criteria for SLE.

Medical records of the study cohort were retrospectively reviewed. Pertinent details regarding their presplenectomy thrombocytopenia treatments, perioperative course, and postsplenectomy follow-up were extracted. Patient's mortality and morbidity were followed to 30 days postsplenectomy in the short term and to their last contact with our institution or to their death in the long term. Patients' platelet counts were followed from postoperative day 2 through day 30 in the short term (early) and then through the last recorded platelet count in the long term (late).

Thrombocytopenia was defined as: 1) medically refractory, if patients did not maintain platelets $\geq 50x10^9$/L for 2 weeks on medical therapy; 2) medically dependent, if patients could not be weaned from medications without a decline of platelets to pretreatment levels; or 3) medically intolerant, if patients had to abandon medical treatments because of severe side effects. Responses of thrombocytopenia to splenectomy were categorized as follows: complete response (CR), platelets $\geq 150 \times 10^9$/L for at least 4 weeks; partial response (PR), platelets $50–149 \times 10^9$/L for at least 4 weeks; or no response (NR), platelets $< 50 \times 10^9$/L at all times after splenectomy. Relapse after splenectomy was defined as a fall of platelet count to less than 50×10^9/L from a prior CR or PR.

RESULTS

Patient Characteristics

Between 1975 and 2001, 7961 adult patients were evaluated for SLE, as defined by criteria from the American Rheumatism Association. During the same period, 6589 open and laparoscopic splenectomies were performed. Of these splenectomies, 75 were performed in patients with SLE. We identified 25 patients in whom splenectomy was performed specifically for thrombocytopenia associated with SLE. This cohort represented 3.1% of all patients with SLE and 3.8% of all splenectomies.

Of these 25 patients, 18 (68%) were women and 8 (32%) were men. The median ages of SLE and thrombocytopenia diagnoses were 31.5 and 34 (range 17–74) years, respectively. The diagnoses were concurrent in 9 (36%) patients. Eleven (44%) patients developed thrombocytopenia at a median of 10.5 (range 0.1–38) years after the diagnosis of SLE. Six (24%) patients had the initial diagnosis of ITP, but after a median of 2.1 (range 0.25–10) years, developed symptoms fulfilling the diagnostic criteria for SLE and were diagnosed with SLE-associated thrombocytopenia.

Presplenectomy Clinical Details and Indications for Splenectomy

At the time of diagnosis of thrombocytopenia, 19 patients (76%) had bleeding symptoms. The median platelet count was 18×10^9/L (range $2–91 \times 10^9$/L). Twenty-three patients (92%) had platelet counts $<50 \times 10^9$/L, and 13 patients (52%) had platelet counts $\leq 20 \times 10^9$/L. Antiplatelet antibodies were not consistently measured. Bone marrow aspirates in 19 patients revealed either increased or normal megakaryocyte counts (Table 1).

All patients were initially managed medically. All patients underwent at least 1 course of corticosteroid therapy. One-third of the 18 patients seen prior to 1990 and all of the 7 patients seen after 1990 received medications in addition to corticosteroids, including immunosuppressive or antineoplastic agents (6 patients), danazol (3 patients), hydroxychloroquine (3 patients), and IVIG (1 patient). The median duration of medical therapy was 6 (range 0–216) months: 4.5 months prior to 1990, and 13 months after 1990.

Despite medical therapy, 8 patients (32%) had persistent bleeding symptoms immediately before splenectomy. Their median platelet count rose to only 36×10^9/L (range $6–165 \times 10^9$/L). Fourteen patients (56%) had platelet counts $\leq 50 \times 10^9$/L, and 6 patients (24%) still had platelet counts $\leq 20 \times 10^9$/L. Overall, 16 patients (64%) were refractory to multiple medical therapies. Five patients (20%) had medically dependent thrombocytopenia. Four patients (16%) experienced intolerable side effects from medical therapy, including osteoporotic fractures, severe diabetes, insomnia, and personality changes. No patient required emergency splenectomy for bleeding.

Operative Details, Operative Mortality and Morbidity, and Immediate Hematological Outcomes

The median age at splenectomy was 39 (range 17–74) years. Splenectomy was performed open in 23 patients who presented prior to 1997 and laparoscopically in the 2 patients

TABLE 1. Presplenectomy Clinical Variables in 25 Patients

Variable	Value	Pre-1990 (n = 18)	1990 and Later (n = 7)
Age	Median (range), yrs		
At diagnosis of SLE	31.5 (17–74)	33 (17–74)	28 (22–63)
At diagnosis of thrombocytopenia	34 (17–74)	34 (17–74)	37 (21–66)
At time of splenectomy	38.5 (17–74)	37 (17–74)	37 (22–68)
Gender	No. (%)		
Male	8 (32%)		
Female	18 (68%)		
Platelet			
At initial diagnosis of thrombocytopenia, median (range), $\times 10^9$/L	18 (2–91)	19 (2–70)	9 (4–91)
Platelet $\leq$ 50, no. (%)	23 (92)	16 (89)	6 (86)
Presplenectomy, median (range), $\times 10^9$/L	36 (6–165)	33 (6–165)	41 (6–117)
Platelet $\leq$ 50, no. (%)	14 (56)	10 (55)	4 (56)
Symptomatic from thrombocytopenia	No. (%)		
At diagnosis	19 (76)		
Symptoms: Petechia, ecchymosis	15		
Mucosal bleed	3		
Epistaxis	6		
Menorrhagia	3		
Presplenectomy	8 (32)		
Symptoms: Petechia, ecchymosis	3		
Mucosal bleed	1		
Epistaxis	2		
Menorrhagia	2		
Other	2		
Bone marrow			
Increased megakaryocytes	12		
Normal megakaryocytes	7		
Not performed	6		
Presplenectomy medical therapy			
Medication, no. (%)			
Corticosteroids only	12 (48)	12	0
1 additional medication	8 (32)	4	4
2 additional medications	3 (12)	1	2
3 additional medications	2 (8)	1	1

SLE, systemic lupus erythematosus.

seen after 1997. Only 1 patient had previous abdominal surgery (left nephrectomy). Only 2 patients had palpable splenomegaly on preoperative physical examination. Median splenic weight was 204 (range 48–764) grams among 22 patients with such data. Median operative blood loss estimate was 300 (range 0–1500) mL in 19 patients. Four (16%) patients with blood losses of 800 to 1500 mL required packed red blood cell transfusions intraoperatively. Eleven (44%) patients received platelet transfusions intraoperatively.

There was no perioperative mortality within 30 days of splenectomy. Six (24%) patients experienced complications. Four were hemorrhagic: gastrointestinal hemorrhage (1), epistaxis (1), abdominal wall ecchymosis (1), and wound hematoma requiring operative evacuation (1). Two were infectious, including severe leukopenia and urinary tract infection. Median hospitalization was 7 (range 4–14) days prior to 1990 and 5 (range 4–10) days after 1990.

On postsplenectomy day 2, hematological responses were complete (CR) in 5 patients, partial (PR) in 14 patients, and none (NR) in 6 patients. By postsplenectomy day 30, CR was seen in 16 patients (64%) and PR in 6 patients (24%), yielding an overall response rate of 88%. Three patients

(12%) remained with persistent severe thrombocytopenia. Two of these 3 had relapsed during this early period, 1 after a transient complete response and another after an initial partial response. Neither was able to rebound their platelets within the early period.

Late Mortality, Morbidity, and Hematological Outcomes and Relapses

Median overall follow-up was 9.5 (range 0–21.7) years from splenectomy. Three patients were lost to long-term follow-up. Nine (36%) patients have died. Most deaths (67%) occurred late, at a median of 11.5 (range 0.6–21.7) years postsplenectomy. Causes of death were cardiopulmonary diseases in 4, bleeding in 1, and unknown in the others. The 1 patient who died of an upper gastrointestinal hemorrhage 7 months postsplenectomy had persistent thrombocytopenia. Significant long-term morbidity occurred in 2 patients. One developed abdominal wall ecchymosis 2 months postsplenectomy associated with a platelet count of 4×10^9/L. The other patient developed meningitis secondary to *S. pneumoniae* 9 years after her splenectomy. She had received pneumococcal vaccination on postoperative day 4.

The long-term hematological outcomes were obtained in all patients for a median of 6.6 (range 0.5–20) years postsplenectomy. Sixteen patients attained partial or complete responses without relapse, yielding a relapse-free response rate of 64%. Eight (32%) of these attained durable remission after splenectomy alone, responding completely (6 patients) or partially (2 patients) without additional medical therapy. The other 8 patients (32%) responded completely (6 patients) or partially (2 patients) to a combination of splenectomy and adjunctive medical therapy.

A total of 9 (36%) patients relapsed and experienced recurrent thrombocytopenia. Two relapsed early (<30 days postsplenectomy) as discussed above. By the last follow-up, one of them was salvaged to CR by subsequent medical therapy. The other did not respond to salvage treatments and was the patient who died of gastrointestinal hemorrhage discussed above. Seven patients relapsed late at a median of 6 (range 0.1–9) years postsplenectomy. Four of these were salvaged to complete (3 patients) or partial (1 patient) responses by the last follow-up, whereas 3 did not respond. Salvage medical therapy consisted of IVIG, hydroxychloroquine, and colchicine in 1 patient each, danazol in 4, and antineoplastic agents in 6. One patient was salvaged after a residual accessory spleen was identified and resected. Thus, aggressive medical therapy and accessory splenectomy salvaged a total of 5 relapsed patients (20%).

Overall, thrombocytopenia was markedly improved in 84% of the patients (64% without relapse and 20% after relapse). Although splenectomy alone attained durable remission in 32% of these patients, it clearly potentiated responses to adjunctive medical agents in the remainder, who were initially refractory, dependent, or intolerant to medical therapy. At the last follow-up, 4 patients (16%) had persistent thrombocytopenia. These had all relapsed and failed salvage. Three of the 4 had died, 1 of bleeding and the others of unknown causes.

DISCUSSION

Only limited information is available on treating thrombocytopenia associated with SLE. Our study further reports the efficacious role of splenectomy for the treatment of thrombocytopenia associated with SLE. We found that splenectomy can be safely performed with minimal mortality and morbidity. Nearly all patients (88%) responded immediately, and 64% sustained their responses without relapse over 6.6 years. Thirty-two percent of the patients attained durable remission by splenectomy alone, whereas an overall 84% improved thrombocytopenia with splenectomy and adjunctive medical therapy.

This study represents one of the largest series of thrombocytopenic patients with SLE who have undergone splenectomy. Additionally, reflecting our practice patterns of a tertiary referral center, the current study also contains one of the most complete and lengthiest follow-ups. Our study cohort represented only 3.1% of all patients with SLE and 3.8% of patients undergoing splenectomy during our institutional study, which highlights the rarity of symptomatic SLE-associated thrombocytopenia. Systemic lupus erythematosus is known to share clinical features with ITP and antiphospholipid syndrome (APS)[26–28]; 3% to 16% of ITP patients may develop SLE.[26,29,30] Lengthy follow-up is thus necessary to accurately exclude patients whose thrombocytopenia is not secondary to SLE and to include patients who may be initially misdiagnosed with ITP. The duration of our follow-up for risk of relapse and late deaths to 6.6 and 9.5 years, respectively, in all except 3 patients likely accurately reflects the uncommon prevalence of this association.

Since the 1980s, numerous medical treatments for patients with thrombocytopenia and SLE have evolved. Patients seen before and after 1990 differ in that the latter group contains fewer patients, received a greater variety of medications, and underwent a longer trial period of medical therapy. Medical therapy resulted in a higher median platelet count (36×10^9/L versus 18×10^9/L), fewer patients with platelets $<50 \times 10^9$/L (56% versus 92%), and fewer patients with bleeding symptoms (76% versus 32%) immediately before splenectomy when compared with at their initial diagnoses. However, a significant portion of the patients responded only transiently (20%) or poorly (64%). Additionally, 16% of the patients were intolerant of the side effects of the immune-modulating medications. Therefore, splenectomy may be an attractive alternative or a necessity in certain patients despite progress in medical therapy.

Previous studies have raised concerns regarding the operative risks in these patients, given their bleeding tendency and underlying multisystem diseases.[31,32] Although operative mortality of 20% has been reported,[10,30] we have shown that splenectomy can be performed safely. Although our perioperative morbidity rate of 24% is comparable to the 20% to 37.5% reported by others,[10,22] no patient in our series had intra-abdominal bleeding, subphrenic abscess, or pancreatitis–complications specific to splenectomy.[33] However, factors that potentially increase the technical difficulty of splenectomy (eg, high patient body mass index, previous abdominal operations, splenomegaly) were absent in most of our patients. Since 1997, laparoscopic splenectomy was performed with minimal blood loss, a short hospital stay, and no complications. With their relative young age, few previous operations, and infrequency of splenomegaly, this patient population is the ideal candidate for laparoscopic splenectomy. The operative mortality and morbidity for this indication will likely continue to decrease with advancement of laparoscopic techniques.

Hematological response to splenectomy was comprehensively assessed in this study by the presence and duration of platelet count response, remission rates, and relapse rates. Novel to the current report, responses to splenectomy alone (ie, durable remission) versus to splenectomy plus adjunctive medical therapy were differentiated, and relapses were followed to their final outcomes. No single previous report had examined all these outcome variables over the long-term. Although reports vary slightly in their definitions of "response," "durable remission," and "relapse," our early response rate of 88%, late relapse-free response rate of 64%, and durable remission rate of 32% exceed those in most previous reports (Table 2). In fact, Hall et al[22] from our institution had reviewed the outcome of splenectomy in 14 patients with SLE between 1960 and 1982. They reported only a 14% remission rate and a 79% relapse rate after a median follow-up of 1.8 years. They concluded that splenectomy was not efficacious for the thrombocytopenia of SLE and recommended exhaustion of alternate treatments before splenectomy due to the low response rate. The reason for their much lower durable remission rate remains unknown. The severity of thrombocytopenia was similar between Hall et al's study and ours, in terms of lowest platelet counts (8×10^9/L versus 8.5×10^9/L), peak presplenectomy platelet counts (70×10^9/L versus 36×10^9/L), and patients with medically refractory disease (71% versus 84%). Differences in overall response and relapse may have resulted from more aggressive medical therapy after postsplenectomy failure or relapse since Hall et al's report. Immunosuppressants such as mycophenolate mofetil (Cellcept®, Roche Laboratories) and antineoplastic agents such as vincristine, vinblastine, cyclophosphamide, and intravenous immunoglobulin were not available or widely used at the time of the previous report. The level and durability of our reported response rates suggest that when splenectomy fails to cure thrombocytopenia alone, it can potentiate patients' response to medical therapy and improve their thrombocytopenia from medically refractory to medically manageable. Our results further challenge Hall et al's conclusion that thrombocytopenia in SLE does not respond to splenectomy in the same fashion as does ITP. Indeed, the response rates are comparable. A recent review of 140 patients with ITP from our institution showed an imme-

TABLE 2. Summary of Hematologic Responses to Splenectomy for Thrombocytopenia of SLE

Author (year)	No. of Patients*	Response (%)†	Durable Remission (%)‡	Persistent Thrombocytopenia§	Relapse (%)	Follow-up (yrs) Median (range)
Homan and Dineen (1978)[10]	10	60	10	0	NA	1 (0–30)
Gruenberg et al (1986)[1]	12	50	17	25	NA	6.1 (0–37)
Hall et al (1985)[22]	14	21	14	57	79	1.8 (0–14)
Jacobs (1986)	6	83	NA	0	17	NA
Coon (1988)[23]	18	78	28	17	6	1.3 (0–15)
Raguin et al (1989)[37]	7	86	28	NA	28	6.6 (NA)
Hakim et al (1998)[26]	8	38	25	0	13	7.1 (2–14)
Mestanza-Peralta et al (1997)[30]	14	92.7	NA	7.1	NA	7.2 (0–32)
Arnal et al (2002)[38]	17	65	NA	41	NA	4.6 (0–30.8)
You et al (current)	25	64 (relapse free) 84 (overall)	32	16	36	9.5 (0–21.7)

SLE, systemic lupus erythematosus; NA, not applicable.
*Includes only SLE patients who underwent splenectomy specifically for thrombocytopenia.
†Platelet count $> 59 \times 10^9$/L at last follow-up;
‡platelet count $> 150 \times 10^9$/L with no medical therapy;
§platelet count $< 50 \times 10^9$/L at last follow-up.

diate complete or partial response rate of 88%, a 1-year relapse-free complete or partial response rate of 81%, and a 1-year relapse rate of 19%.[34] Whether splenectomy for thrombocytopenia with SLE should become the standard as has splenectomy for ITP is debatable, but its role must clearly be considered. Further study of splenectomy for SLE thrombocytopenia will help define its contribution to the successful management.

Identifying reliable predictors of poor response to splenectomy was difficult in this study. First, early failure did not predict late failure. One early nonresponder who underwent splenectomy for medically refractory thrombocytopenia maintained complete response for 3.1 years on adjunctive low-dose corticosteroid. Although splenectomy was not sufficient treatment of her thrombocytopenia, it made her disease medically responsive and manageable. Secondly, adequate early response did not preclude relapse, but relapse did not predict long-term failure. One patient had an early partial response but relapsed within 30 days of splenectomy. Her relapse was refractory to any salvage treatment, and she eventually died of an upper gastrointestinal tract hemorrhage. Three other patients had early partial or complete responses but relapsed at 3, 22, and 28 months postsplenectomy. Although these relapsed patients also failed maximal salvage medical therapy, 5 other patients who relapsed were successfully salvaged, by either medical therapy or accessory splenectomy, to PR or CR at late follow-up. Finally, no other demographic, preoperative, or intraoperative factors were correlated significantly to ultimate failure. Our overall study population is too small to allow meaningful univariant or multivariant analyses to identify predictors of hematologic response to splenectomy. Moreover, meta-analysis of the literature is also precluded because of small retrospective studies reported to date. Although a multicenter study may provide sufficient data necessary to identify prognostic factors for the selection of patients who may benefit the most from splenectomy, further corroboration of outcome similar to ours may better define the clinical utility of splenectomy.

Lastly, by improving thrombocytopenia, splenectomy not only increases platelet counts, but also enhances the quality of life in these patients. In our study, thrombocytopenia of SLE affected predominantly women in early adulthood. Their life span was significantly shortened to a median age of 56 years at death, by cardiopulmonary complications of SLE rather than bleeding secondary to thrombocytopenia. Cardiopulmonary and renal complications are known major predictors of poor survival in patients with lupus.[2,35,36] Although our overall mortality of 36% exceeds that in previous reports (6–16%),[1,10,23,30,37] this difference likely relates to our study size and median duration of follow-up (9.5 years). Splenectomy did not adversely affect late mortality. In addition, bleeding diatheses complicated these shortened life spans. Splenectomy reduced these complications from 76% of the patients preoperatively to only 20% (4 early and 1 late) postoperatively. Only 1 patient had a major infection, fulminant pneumococcal meningitis, despite prophylactic postsplenectomy vaccinations. Furthermore, 32% of patients were freed from side effects of medical therapy, as they required no adjunctive postsplenectomy therapy. The remaining patients who remained on medical treatments for their SLE or thrombocytopenia typically required fewer varieties of medications and lower doses, as splenectomy likely potentiated their response to medical therapy. Lastly, although we did not specifically compare the disease severity of SLE before and after splenectomy, several studies have shown that splenectomy does not exacerbate SLE.[1,26,31,38] Therefore, another rationale for splenectomy early in a patient's treatment may be to improve their quality of life. Future studies utilizing standardized instruments to measure patients' quality of life before and after splenectomy will help elucidate this issue.

In summary, we demonstrate in this large institutional experience that thrombocytopenia associated with SLE should not be a contraindication to splenectomy. Importantly, hematological outcomes were comprehensively assessed in terms of durable remission, response, and relapse over a lengthy follow-up period. Splenectomy has the potential to provide long-term control of thrombocytopenia in SLE patients who do not respond to or cannot be weaned off corticosteroids. The procedure is safe, with low mortality and morbidity. Nearly two-thirds of patients improved thrombocytopenia in the long-term without relapse, either by splenectomy alone (one-third) or by splenectomy and adjunctive medical therapy (one-third). Though relapses may occur late at 6 years after splenectomy, over half of the relapses can be salvaged medically even if patients were refractory to such therapy prior to splenectomy. Overall, splenectomy improved thrombocytopenia in 84% of the patients, either alone or by potentiating responses to adjunctive or salvage medical therapy. No perioperative factors reliably identify patients who may have persistent thrombocytopenia in the long term.

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